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MEDICAL MANAGEMENT IN PEDIATRIC SURGERY

Special Report

Frederic G. Burke, M.D.

Infants and children are frequently regarded as miniature men and women both in the general practice of medicine and surgery. Consideration of the factors of immaturity, growth, and development attest to the incorrectness of this viewpoint. During the first few years of life the patient is characterized by markedly immature but very active physiologic processes and rapid metabolism necessitated by the acceleration of growth. There is a particular instability of the mechanisms that control water, acid-base, and nitrogen balance. The younger and more immature the infant or child, the more rapid the extensive changes that occur in physiochemical disturbances of the normal balance of tissue fluids and the greater the loss of circulating blood cells in the plasma. Normally the quantity of circulating plasma is remarkably constant representing about 5 per cent of the body weight while interstitial fluids constitute 15 per cent and the intracellular fluids, 50 per cent of the body fluid. Under the conditions of disease, changes in the volume and composition of blood plasma closely reflect the changes of the fluids in other compartments. There are many mechanisms with a remarkably standard pattern under abnormal states which are activated to conserve body fluids and minimize the loss of water. These changes are characterized primarily by shifts in the concentration of the various water holding salts within their respective compartments. Since so many surgical disorders result in anemia, dehydration, acidosis, and alkalosis the pediatric surgeon and the pediatrician must reckon with these changes in the management of a sick child if optimal results are to be obtained. As important as these factors are in the preparation of the adult for surgical procedures, the lability and immaturity of these balancing mechanisms in children make their adjustment of paramount importance.

Physiologic salt solution is frequently used in the fluid replacement therapy of children, and if the kidneys are functioning normally it helps correct alkalosis or acidosis. Excessive amounts, particularly in the presence of decreased kidney function, will lead to edema on the basis of sodium retention. The various modifications of Ringer's solution with magnesium, calcium, and potassium added are to be preferred. Hartmann's solution contains sodium lactate in addition to the other ions of Ringer's solution and it is more effective in correcting acid-base imbalance associated with dehydration. Glucose should be used in conjunction with Hartmann's-Ringer's solution when ketosis is present and it is well tolerated and utilized

as a 5 per cent solution given either intravenously or subcutaneously. Five per cent glucose with Hartmann's solution may be used to good advantage in helping to restore the mineral and caloric imbalance in pre- and post-operative care and is probably the safest parenteral fluid to be employed when facilities for frequent carbon dioxide combining power determinations of the blood are not available. When possible, the oral route for hydration is by far to be preferred particularly in reference to caloric restoration. Fluids by rectum such as retention enemas are similarly quite effective in restoring fluid balance.

The effects of hypoproteinemia are many and well-known. The hypoproteinemic child is more susceptible to infection and when infection supervenes, the course is apt to be stormy and protracted. Children who prior to their development of a surgical disorder were suffering from a protein deficiency develop an aggravation of this condition during the disease process and may succumb even in the face of bacteriologic control by antibiotics because of the severe decrease of tissue and plasma proteins. Furthermore, he is more susceptible to shock and post-operatively may suffer from delayed rate of healing and his chances of developing a post-operative adynamic ileus is increased. Stomal difficulties, chronic anemia and intestinal obstruction are more likely to occur when the blood proteins are lowered. The development of ascites and the effects of hypoproteinemia in hemorrhage, burns, peritonitis, and obstruction are well known.

When possible, protein should be supplied by mouth. In infancy when the intake falls below 2.2 grams per kilogram of body weight, a negative balance is to be expected.

Amino acid solutions are frequently used to control and maintain nitrogen equilibrium and are usually given as a 3½ per cent solution with dextrose (amigen) either subcutaneously or intravenously to children who for one reason or another cannot take adequate amounts of protein by mouth. They are particularly useful when the alimentary tract is obstructed as in atresia, pyloric stenosis, intussusception, volvulus, peritonitis, or when the intestinal tract should be kept at rest during the post-operative course of abdominal surgery. Amino acids are essential in the tissue synthesis during convalescence from these conditions. The value of plasma in restoring circulating volume while raising the intravascular osmotic pressure is well known in the management of the adult surgical patient and is equally valuable in children.

Blood transfusions are indicated for the replacement of blood lost by hemorrhage and are considered essential when the hemoglobin content is below 10 milligrams per cent. The frequently noted beneficial effects of small repeated blood transfusions in the post-operative course of children justify the use of blood even though the hemoglobin and red blood cell

counts have slightly higher values. This effect has been attributed at least in part to naturally occurring antibacterial factors and to the carbonic anhydrase contained in adult blood in addition to its immediate value as a source of protein and oxygen carrying properties.

Vitamin K should be administered to the newborn before and after surgical procedures to circumvent hemorrhagic diathesis and attention should be paid to the calcium and vitamin C intake particularly in early infancy.

Because of the ever present danger of concurrent or post-operative infection, the use of antibiotic and chemotherapeutic agents just before and following any major surgical procedure is advisable. Penicillin, sulfadiazine, aureomycin, and chloromycetin with their wide anti-microbial range are the drugs of choice either alone or in combination and adequate peripheral blood levels should be obtained when time permits before any potentially "dirty surgery" is done and maintained for an adequate post-operative period. Crystalline penicillin in large doses (100,000 to 300,000 units every three to four hours) is used in infants and children when a susceptible infection is known to be present. A depo crystallin procaine penicillin in doses of 300,000 units every twelve hours is considered adequate as a prophylactic post-operative measure, and has the additional virtue of causing less pain and bother. Sodium sulfadiazine, particularly in bowel surgery, may be given as a 5 per cent solution or more diluted subcutaneously—adequate hydration must be assured and blood levels checked when sulfones are employed, for obvious reasons. The use of sulfones and Penicillin have reduced to a minimum the hazardous post-operative complication of respiratory and skin infections. Pre-operative reduction of bowel flora may be effectively accomplished in selected cases by the use of sulfasuxadine, aureomycin, chloromycetin, or polymyxin. Aureomycin intravenously or chloromycetin by rectum may prove to be a very helpful effective measure in the treatment of peritonitis since their anti-microbial range covers the host of probable invading bacteria.

Morphine as pre-operative preparation has been much maligned in pediatric practice and the dangers have perhaps been unduly emphasized. In small doses effective relaxation is possible with a safety margin that is not too narrow. However, the use of morphine in general is contraindicated because of difficulty in obtaining graduated doses, the ever present danger of central respiratory inhibition due to the possibility of using a greater dosage than is necessary, and because there are several other safer drugs available that accomplish the same purpose. In infants, pre-operative sedation is not necessary. In older children barbiturates are quite effective and have the virtue of a wider range of safety. Morphine should be reserved for the more apprehensive child. Where possible, however, because of the

constant danger of psychic trauma, pre-operative sedation in older children is to be strongly recommended. One of the most important points in post-operative management is relief of pain. While apprehension and loneliness may be treated with barbiturates real pain is best relieved by morphine.

Conservation of body heat in young infants during the operative procedure is a point which cannot be over-emphasized. The use of an electric heating pad or hot water bottles around the infant's body particularly during prolonged operations reduces the chances of shock and minimizes the danger of post-operative infection. Oxygen therapy and measures to maintain a normal body temperature are important supportive procedures as are suction facilities for the nose, throat, and stomach during the period of time immediately after the operation.

These are the general points in the management of the young surgical patient and in each specific condition any particular phase may assume peculiar significance. The best pre-operative preparation in the world is a poor substitute for early and accurate diagnosis. For example, in pyloric stenosis, the relative safety and success of surgical correction is attested to by its general acceptance as the method of choice for the treatment of this condition. In adequate hands, the mortality attendant to this operation should be no higher than 1 per cent. Consideration of the physio-chemical equilibrium and fluid balance of these infants before and after operation would promise to further reduce this mortality to an even lower minimum. In no instance should an infant be operated upon for pyloric stenosis without satisfactory pre-operative preparation. This consists of adequate hydration and correction of any existing anemia by blood transfusion and the correction of alkalosis by the parenteral administration of physiologic saline solution and glucose. Probably the two most important factors responsible even for this mortality are the failure to adequately prepare these patients for operation and failure to institute surgical procedures early. It is well known that severe alkalosis can result in generalized convulsions and in fatal depression of respiration in the small infant. The tetany that results from this alkalosis should be relieved by intravenous administration of calcium gluconate. Fluid and acid-base balance can nearly always be restored by the intravenous administration of salt solution. Surgical correction of pyloric stenosis is never an acute emergency and restoration of hemeostasis and caloric equilibrium are essential to its successful outcome. Because these infants are actually in a state of starvation, external heat should be constantly applied before, during, and after operation. Blood should be given intravenously immediately after recovery from the anesthetic. Formulas should be withheld for at least twelve hours because of the frequency of post-operative vomiting and at that time half strength breast milk or a weak acidified evaporated milk formula should be employed in small amounts at three to four hour intervals.

Of considerable importance in elective pediatric surgery is the selection of the time to operate. While the location and nature of the defect that clearly indicates the necessity of prompt surgery offers no problem in timing, there exists a difference of opinion as to when to operate when conditions exist that apparently do not offer an immediate threat to life. An example is the inguinal hernia. Most textbooks have elaborate diagrams of ingenious devices calculated to truss the anatomical weak point in the inguinal region. We personally have had no success in the use of trusses and believe they should be discontinued except in the rare case. There is a very bad psychologically deterrant effect upon the young mother who lives in constant fear of strangulation of the hernia which has its effect not only on the parents but on the infant. Too often these infants are pampered and given everything they want or think they want to prevent them from crying, the parents appreciating that the increased intra-abdominal pressure resulting from crying causes the hernia to descend. While an occasional inguinal hernia will spontaneously heal during the first year it is very doubtful that any inguinal hernia will heal spontaneously after the first year and even if incarceration has not occurred before the twelfth month, at that time surgical repair should be instituted. If incarceration occurs at any time after birth, surgical repair, not a truss, is the correct management.

In many instances surgical correction constitutes the conservative management and the earlier such correction is instituted the better the effect observed in the child as a whole. For example, the hare lip should be repaired in the first few days of life and cleft palate within two years, before phonation has developed. Cardiothoracic, urologic, and orthopedic defects all offer specific problems in timing. Consideration of the peculiar factors of growth and development that characterize the infant and child help in the selection of the time for surgical correction.

SUMMARY

In summary of this general outline of principles of the medical management of the infant or child who is a candidate for a surgical operation, it is well to keep in mind that the operation represents only one specific phase in a treatment regime that does not lose sight of the patient as a whole. As important as the good surgical technique is to the successful outcome, failure to give keen attention to medical factors offers a serious threat to good results.

These factors are:

1. Hydration. This consists of the administration of parenteral fluids if the oral route is not available and also with particular reference to the selection of the correct fluid, route, rate, and technique.
2. Restoration and maintenance of caloric, nitrogen and acid-base balance.

3. Measures to conserve body heat.
4. Relief of apprehension, pain and prevention of psychic trauma by the judicious use of barbiturates and morphine.
5. The restoration of a normal blood picture by the use of small, repeated blood transfusions.
6. Measures calculated to control and prevent infection by suitable antibiotic and chemotherapeutic agents.
7. Symptomatic and supportive therapy as indicated by the particular case, including the use of oxygen and suction facilities for nose, throat, and stomach.
8. With consideration of the factors of growth, development to help in the selection of the time for elective surgery.

TYPHOID FEVER TREATED WITH CHLOROMYCETIN®

Case Report No. 177

William Orr, M.D.

C. M., a three year old colored female, was admitted to Children's Hospital on April 29, 1949 with a chief complaint of spiking fever of two weeks' duration. The only other associated complaint was a mild, generalized abdominal pain. She vomited once two days before admission, but there had been no diarrhea. A physician had treated her with sulfadiazine at home without apparent benefit. Two days prior to admission, the patient was seen in the clinic and procaine penicillin in a dosage of 300,000 units was given intramuscularly. At this time blood was drawn for a Widal test and showed an agglutination with "O" and "H" antigen in dilutions of 1:320 and 1:160 respectively. She was admitted with a provisional diagnosis of typhoid fever.

Family history revealed that an older sister had had a positive blood culture for E. Typhosa two years previously. She had been hospitalized and had made an uneventful recovery. A maternal aunt at that time was found to be a typhoid carrier. The aunt moved from the household two years ago, but visited the family occasionally during this interval.

The patient's past history was negative. She was immunized against diphtheria and smallpox, but had received no typhoid vaccine.

The admission physical examination revealed a well developed and well nourished female. She was lethargic and irritable, but did not appear acutely ill. The temperature was 102.0 F. and the pulse rate 120. She had a slight pharyngitis and a soft systolic murmur at the pulmonic area. The abdomen was moderately distended and the liver was palpable 5 centimeters below the right costal margin. The spleen was not palpable. There were no rose spots. The inguinal nodes were shotty, but no other lymphadenopathy was present.

Hemogram on admission showed a hemoglobin of 9 grams with 3,000,000 red blood cells. The white cell count was 14,200 with 45 per cent neutrophiles, 54 per cent lymphocytes and 1 per cent monocytes. Urinalysis was negative. Daily stool cultures were persistently negative for typhoid organisms and four consecutive blood cultures were also negative. Five consecutive Widal agglutinations obtained during hospitalization were positive in dilutions of 1:320 for both "O" and "H" antigens.

After admission the patient continued to have a daily temperature of 101.0 F. On the third day, the temperature rose to 104.0 F. In view of the positive Widal tests suggestive of typhoid fever, treatment with chloro-

mycetin® was begun on May 2, 1949. An initial dose of 750 milligrams and then 500 milligrams every three hours was given orally. This was continued for seven days and then reduced to 250 milligrams every three hours for four additional days. Two days after chloromycetin® was started, the temperature had returned to normal and remained so for the next twelve days. The medication was well tolerated and produced no untoward symptoms. After the fifth hospital day, the blood count showed a mild but persistent neutropenia, the average leukocyte count being 6,000 with 31 per cent neutrophiles and 69 per cent lymphocytes. Chloromycetin® blood levels ranged between 45 to 80 micrograms per cubic centimeter. Medication was discontinued on May 13, 1949 after eleven days of treatment. Four days later on May 17, the patient was discharged.

The patient was readmitted to the hospital two weeks later with the following interval history. She had been well for about one week after discharge, and then began to have a low grade fever which soon began to spike. There was no vomiting or diarrhea, but her appetite was poor. She complained of generalized aching. Three days before admission, a rash appeared first on her arms and legs and then on her abdomen and chest.

Physical examination on this admission revealed a moderately ill, dehydrated patient with a generalized, discrete, papular skin eruption. The papules measured 1 to 2 millimeters in diameter. The lips and mucous membranes were dry and had ulcerated areas resembling a Vincent's infection. The abdomen was slightly distended, and the spleen was just palpable. The temperature was 104.2 F., pulse rate, 190 per minute, and the respiratory rate 40 per minute.

Laboratory examination revealed a stool culture positive for Eberthella Typhosa. A Widal test showed a positive agglutination for "O" antigen in a dilution of 1:320, and "H" antigen in a dilution of 1:80. Urinalyses, blood cultures, and subsequent stool cultures during hospitalization were negative. Hemogram on admission revealed a hemoglobin of 9 grams with 3,500,000 red blood cells; the white cell count was 9,000 with 40 per cent neutrophiles and 60 per cent lymphocytes.

On the fifth day after readmission when it had become apparent that the patient had a relapse of typhoid fever, chloromycetin® was started. The initial dose was 750 milligrams and then 500 milligrams every three hours thereafter. Within twelve hours, the temperature returned to normal and remainded so during the remainder of the hospital stay. After nine days the dosage was reduced to 250 milligrams every four hours. The patient's general condition improved rapidly and the hospital course was uneventful after institution of chloromycetin® therapy. Chloromycetin® was given for sixteen consecutive days. The patient was discharged on the twenty-third hospital day.

DISCUSSION

Sidney Ross, M.D.: The efficacy of chloromycetin® in the treatment of typhoid fever has been unequivocally established. Since the initial publication of Woodward et al[1], other confirmatory observations have been reported[2]. Our own experience with chloromycetin® in the treatment of typhoid fever pertains chiefly to its use in the pediatric age group. During the past year we have treated ten cases of typhoid fever with chloromycetin® employing an initial dose of 50 milligrams per kilogram of body weight followed by 25 to 30 milligrams per kilogram every three to four hours for an average of fourteen days. In seven of the ten cases there was a prompt drop in temperature within two to four days after initiation of treatment together with a striking clinical improvement. In one case of typhoid peritonitis, no salutary effect was observed and the patient went progressively downhill and died within four days. In one case of uncomplicated typhoid fever, no favorable effect of the drug was noted in spite of the administration of large doses. The temperature remained elevated for twenty-one days, and the course usually noted in an untreated case of typhoid fever ensued. The explanation of the complete lack of response to therapy in the latter case was difficult to determine. In our series positive blood and stool cultures usually became negative within twenty-four to forty-eight hours after initiation of therapy. Chloromycetin® levels obtained in each patient at frequent intervals during treatment ranged between 15 and 80 micrograms per cubic centimeter.

The chief interest of the present case lies in the fact that the patient showed a definite relapse within two weeks after discontinuation of an adequate course of chloromycetin® therapy. Smadel[3] in a discussion of the relation of relapses in typhoid fever to the duration of chloromycetin therapy indicated that in a series of thirteen patients, a clinical relapse occurred in approximately 50 per cent of the cases when the average duration of therapy was eight days or less. However, in thirty-one other patients treated from nine to twenty-three days, not a single relapse occurred. They concluded that chloromycetin® should be administered in adequate amounts for more than eight days to patients acutely ill with typhoid if relapses of the diseases are to be avoided, and similarly pointed out that there was no advantage to continuing treatment for more than fourteen days. However, in the present case, the drug was administered in good dosage for eleven days during the first course of therapy with blood levels ranging between 45 and 80 micrograms per cubic centimeter. In view of the prompt initial response to therapy, it was not considered necessary to continue the drug beyond this interval. However, within two weeks after discontinuation of chloromycetin® a definite relapse occurred with the appearance for the first time of a positive stool culture. When therapy was started again, a

prompt sterilization of the intestinal tract occurred together with a rapid return of the temperature to normal and the patient made an uneventful recovery. During this second course of therapy, the drug was continued for sixteen days. Follow-up stool cultures during the next two months have remained consistently negative.

Smadel et al[3] recommend that chloromycetin® therapy be continued for eleven to fourteen days in typhoid fever in order to avoid relapses. In the large majority of patients, this should suffice. However, there will undoubtedly be an occasional instance where relapses will occur in spite of adequate therapy administered for the recommended interval.

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ACUTE ANTERIOR POLIOMYELITIS

Case Report No. 178

Joseph M. LoPresti, M.D. G. L. H. 5822–49

G. L. H., a seventeen year old white male was admitted to the St. Vincent's Infirmary in Little Rock, Arkansas on August 9, 1949.

The history revealed that four days prior to admission fever, headache, nausea and vomiting, and nuchal rigidity developed. Three days later weakness of the left upper extremity became manifest. At the same time the patient had some difficulty in swallowing. On the day of admission irregular, rapid respirations were noted. Throughout the present illness, the food and fluid intake had been poor. Constipation and urinary retention had been present almost from the onset. A few hours before hospitalization, the patient had become extremely nervous and apprehensive.

Two interesting facts were elicited from the past history:

1. The patient had engaged in violent exercise on the day that he became ill.
2. An intimate playmate had been hospitalized with the diagnosis of acute anterior poliomyelitis about one week prior to this patient's admission.

The admission physical examination revealed the temperature to be 101.4 F., respirations were 48 per minute, and the pulse rate was 80 per minute. The blood pressure was 160/84. The patient was an extremely well-developed and nourished white male who was acutely ill. The facies was quite flushed and circumoral pallor was present. He was very apprehensive and overly-alert. Speech was rapid, blurred, and nasal in character while commands were obeyed with startling rapidity. When fluid was offered, the patient could not swallow and it was regurgitated through the nose. Although the thoracic respiratory excursion appeared adequate, the respiratory rhythm was grossly irregular and characterized by alternating periods of dyspnea and apnea. The pharynx was moderately injected and the accumulation of a moderate amount of thin mucus was noted. However, the patient was able to cough this up without difficulty. Marked nuchal rigidity and stiffness of the back were present. The bladder was distended and reached the level of the umbilicus. The Kernig and Brudzinski signs were positive and moderate hamstring spasm was present bilaterally. The left biceps reflex was diminished; all other reflexes were physiologic. There

Editor's note: Dr. LoPresti is a member of the house staff at The Children's Hospital of D. C., and was sent by the National Foundation for Infantile Paralysis to serve as pediatrician in the epidemic area of Arkansas.

was marked weakness of the muscle groups around the left shoulder girdle, particularly the deltoid, and a generalized weakness of all of the muscle groups of the left upper extremity.

The admission spinal fluid obtained from lumbar puncture was reported as showing 110 cells per cubic millimeter of which 60 per cent were polymorphonuclear forms and 40 per cent were lymphocytes. The sugar content was normal and the protein was elevated to 60 milligrams per cent. The hemogram revealed 15.0 grams of hemoglobin and 4,300,000 erythrocytes. The leukocytes numbered 7,700 with a differential of 76 per cent polymorphonuclears and 24 per cent lymphocytes. The urinalysis was normal except for an acetonuria of 4 plus and a concentration of 1.033.

Eight hours after admission, the patient began to complain of tightness in the chest and girdle-like pain in the region of both diaphragms. Examination revealed a respiratory rate of 58 per minute, flaring of the alae nasi, and limitation of the thoracic excursion particularly on the left side. He was immediately placed in a Drinker respirator. For the next two days, the temperature fluctuated between 101.0 F. and 102.0 F. Although the amount of pharyngeal secretions increased, the patient was able to cough them up adequately. He accommodated well with the respirator and became less apprehensive. A hypertension of 160 to 170 systolic, and 90 to 100 diastolic persisted. However, at this time, the development of weakness of both lower extremities was noted and it was evident that the infectious process was still active and progressive. By the afternoon of the third day following hospitalization, the picture of a full-blown encephalitis had become manifest. The patient lapsed into semi-coma and became markedly disorientated. Auditory and visual hallucinations were present. The febrile course remained unaltered. At this point, the intramuscular administration of a 50 per cent magnesium sulfate solution was instituted. The dose administered was 4.0 cubic centimeters every four hours, approximately 0.05 cubic centimeters per kilogram of body weight. Blood pressure readings were recorded just prior to each dose of magnesium sulfate and one hour subsequent to its administration. At the onset of therapy the blood pressure was 170/115, the pulse rate was 120 per minute, and the temperature was 101.4 F. One hour following the first dose of magnesium sulfate, the blood pressure was 140/100 and the pulse rate had dropped to 80 per minute. Subsequently the blood pressure and pulse rate remained stationary at 140/90 and 90 per minute respectively. After the third dose of magnesium sulfate, a change in the patient's sensorium was noted. He became more alert, began to talk rationally, and subjectively appeared improved. At the same time, the temperature became normal for the first time since admission to the hospital. His hospital course was then one of sustained and continued improvement. Altogether, he received fourteen doses of magnesium sulfate.

One week after admission, fluids could be tolerated by mouth and by the seventeenth day of hospitalization, the patient was entirely out of the respirator.

During the entire febrile course urinary retention was a major problem. This was adequately and efficaciously combatted by the subcutaneous administration of 2.5 milligrams of furmethide. This urinary stimulant caused complete emptying of the urinary bladder within fifteen minutes of its administration.

DISCUSSION

The case herein presented is deemed worthy of reporting because the protean features indicate many interesting prognostic and fundamental facts. From the very day of admission it was almost possible to predict what would happen to the patient. Four findings made the prognosis a grave and guarded one:

1. The presence of fever indicated an infective process which was still active. Therefore, the progression of paralysis was a definite possibility.
2. The presence of hypertension signified an involvement of the circulatory center by the infectious agent. In this type of bulbar poliomyelitis, the mortality rate is high as the patient often develops sudden peripheral collapse.
3. The grossly irregular respiratory rate was indicative of respiratory center involvement. The presence of other bulbar symptoms, i.e., hypertension, dysphagia, and nasal voice signified widespread bulbar involvement.
4. The involvement of the left upper extremity indicated imminent intercostal and/or diaphragmatic paralysis. Subsequent events bore out this observation. In a short time it was necessary to utilize the Drinker respirator because of the involvement of the muscles of respiration.

With these factors in mind, the therapy for bulbar poliomyelitis recommended by Wilson[1] was instituted. Basically this routine includes six fundamental features: 1. Nothing by mouth; 2. The maintenance of fluid balance with parenteral fluids, plasma, and blood; 3. Parenteral penicillin as a prophylactic measure; 4. Nasal oxygen; 5. Trendellenberg position to utilize gravity in draining the pharynx; and 6. Suction of the pharynx as necessary. In the same paper Wilson points out that the respirator is rarely effective in any of the situations which result from bulbar poliomyelitis while it is a life-saving measure in high spinal cord paralysis. Therefore, any patient with involvement of the intercostal muscles and/or the diaphragm is a candidate for the respirator even if such a patient has concomitant bulbar involvement.

Another fundamental factor is illustrated by the case presented in this paper. Russell[2] in England has pointed out the relationship of physical activity in the preparalytic stage of poliomyelitis and the severity of the paralysis. There can be no doubt that severe exertion early in the course of poliomyelitis has a profound effect on the subsequent course of the disease. Physical activity results in more severe and more extensive paralysis. Our patient engaged in a rather strenuous game of baseball on the day that he became ill. It is probable that this exertion accounted in part for the severe nature of his disease. Quite frequently in poliomyelitis, there is a three or four day asymptomatic period following the nonspecific prodromal phase of the disease. This period when the patient is symptom-free is misleading and dangerous. In an area where poliomyelitis is epidemic or endemic, a patient who presents the prodromal symptoms consistent with early poliomyelitis should be kept in bed and at rest for a safe period of time, preferably seven to ten days.

The use of parenteral magnesium sulfate in the case presented was a therapeutic measure instituted as a last resort. It lends itself to a considerable amount of speculation. While it is recognized that a high probability of coincidence exists, it still stands that the patient's improvement occurred concomitantly with the administration of the magnesium sulfate. It is possible that the drug exerted a beneficial therapeutic effect. A number of workers[3, 4, 5] have investigated the physiologic action of the magnesium ion. It is primarily a central nervous system depressant. On introduction into the blood stream, the first and most constant result is a fall in the blood pressure. The blood vessels are markedly dilated. At high enough concentration a failure of natural respiration and cardiac arrest is produced. The hypertension which so often accompanies glomerulonephritis in childhood can be relieved by magnesium sulfate, a therapeutic measure introduced by Blackfan[6]. Rubin and Rapoport[7] have demonstrated that the cerebral symptoms in acute glomerulonephritis appear following an acute rise in blood pressure and are in greatest probability due to cerebral ischemia resulting from the generalized vasospasm which is also the cause of the hypertension. Measures designed to reduce the vasospasm, i.e., parenteral magnesium sulfate, result in a disappearance of the cerebral symptoms and a reduction in the blood pressure. Are the hypertension and cerebral symptoms which accompany some cases of bulbar poliomyelitis related to cerebral ischemia and vasospasm? If so, then the intramuscular administration of hypertonic magnesium sulfate may be a valuable therapeutic adjunct in the management of such cases. Certainly, it is worthy of further trial and investigation.

No discussion of poliomyelitis would be complete without a presentation of the brilliant clinical observations of Baker[8] of Minnesota. On the basis

of a detailed study of the symptomatology observed during life and supported by pathological findings in fatal cases, he has divided bulbar poliomyelitis into five groups:

1. The upper cranial nerve nuclei group includes involvement of the third, fifth, sixth, seventh, and eighth cranial nerves. Such involvement holds no threat to the life of the patient. Its importance rests in the fact that they should make the physician alert to the possibility of the implication of more vital centers.
2. The lower cranial nerve nuclei group. By far the most frequently involved cranial nerve nucleus in bulbar poliomyelitis is the tenth. These patients have a weakness or paralysis of the soft palate, the pharynx, and the vocal cords. Their initial complaint is a nasal twang to the voice, hoarseness, increased accumulation of oropharyngeal secretions. An occasional patient is unable to talk and laryngeal stridor may be present. Such lesions furnish an immediate threat to the life of the patient primarily because of the possibility of occlusion of the upper respiratory tract by:
   a. Accumulation of vomitus or saliva in the pharynx,
   b. Closure of the glottis in reflex spasm, or
   c. Abductor paralysis of the vocal cords with their approximation in the midline.
3. The respiratory center group. Most of these patients will show some cranial nerve involvement. The outstanding symptoms are respiratory, characterized by the development of irregularities of rhythm and depth of respiration. The respirations tend to be shallow and there are prolonged intervals between inspirations. These respiratory symptoms occur despite strong intercostal muscles and diaphragm. The patient dies of progressive respiratory failure.
4. The circulatory center group. Such involvement is most frequently observed in the more severe cases of bulbar poliomyelitis in which the cranial nerve nuclei and the respiratory centers are predominantly involved. When the symptoms of circulatory involvement supervene in any form of bulbar poliomyelitis, the prognosis is extremely grave. The patients have a dusky red, flushed, florid appearance. The lips are a deep cherry red. The pulse is very rapid and often irregular, ranging between 150 and 200. The patient dies in peripheral circulatory collapse.
5. The encephalitic group. Encephalitic symptoms occur commonly in bulbar poliomyelitis and appear within the first few days of the illness. Whether these symptoms are secondary to cerebral hypoxia or to direct involvement of the brain by the virus is an unestablished and debatable point.

More than half of the patients with bulbar poliomyelitis have an involvement of the cranial nerve nuclei.

Seldom is there an involvement which permits a clear cut classification into one of the groups. Most frequently there is a dove-tailing of groups with one group predominating.

SUMMARY

1. An interesting case of acute anterior poliomyelitis has been presented.

2. Some fundamental principles in the prognosis and progress of poliomyelitis have been indicated.

3. Speculation as to the value of magnesium sulfate as a therapeutic measure in certain forms of poliomyelitis has been made.

4. The present classification of bulbar poliomyelitis has been given.

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SICKLE CELL ANEMIA

Special Report

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Sickle cell anemia is a true hemolytic anemia first described by Merrick in 1910. Huck of Johns Hopkins demonstrated the sickling phenomenon and showed that it was an inherent trait of the red cell rather than one of the plasma. The disease is transmitted according to the Mendelian Law of Heredity and is limited exclusively to the Negro race, although a few cases have been described in other races mainly in persons of Mediterranean origin.

Many theories regarding the factors producing the disease have been recorded. Some of these are:

1. An hereditary bone marrow defect causing the production of these cells.
2. Changes in the plasma oxygen tension with sickling resulting from a lowering of the tension.
3. A variation in the hydrogen ion concentration of the blood may be responsible because acid media enhance sickling.
4. Sickling may be due to a structural weakness of the cellular network of the erythrocytes.
5. A disturbance in the blood plasma in infections may be the factor which precipitates the onset of sickling.
6. An overactivity of the spleen has yet to be proved as a cause of sickling.

Sickle cell disease has been an interesting and frequently encountered problem at The Children's Hospital mainly because of the many Negro admissions. In the ten year period from 1938 to 1949 we were able to collect fifty-eight cases with a definite diagnosis of sickle cell anemia. Of these, thirty-two were Negro males and twenty-six were Negro females. They required a total of two hundred-twenty admissions to the hospital representing 2,727 hospital days. This represents an average of 3.8 admissions or 47 hospital days per patient.

The signs and symptoms of sickle cell anemia are so protean in nature that the diagnosis is often extremely difficult to establish. The general picture is one of poor health, the patient often appearing tired, poorly developed, and undernourished. There are usually recurrent attacks of weakness, tachycardia, prostration, and even circulatory collapse. Many of the

symptoms may be referable to disturbances of the gastro-intestinal tract, including generalized or upper abdominal pain, nausea, and vomiting. Joint pains resembling arthritis are often present. During an acute attack the disease may be confused with an acute abdominal emergency or with an arthritic syndrome. The upper abdominal pain is thought to be due to infarctions of the spleen. Jaundice is frequently noted and pallor is usually present. There may be slight generalized lymphadenopathy. Bone changes are sometimes encountered and there may be an increase in the pulse and respiratory rates and an elevated temperature.

In this study the average age of onset of clinical manifestations was 3.2 years. The youngest of these was seventeen days and the oldest, eleven years. The seventeen day old infant is, to our knowledge, the youngest patient on record with a hemolytic crisis of sickle cell anemia. Cases grouped according to age were as follows:

Age	*Number of cases*
0 to 6 mos.	8
6 mos. to 1 yr.	9
1 yr. to 2 yrs.	15
2 yrs. to 4 yrs.	13
4 yrs. to 7 yrs.	7
7 yrs. to 11 yrs.	6

Forty-four patients or 77.6 per cent had their onset in the first four years of life. Thirty-two patients or 55.2 per cent were two years or under.

Initial Symptoms in 58 Cases of Sickle Cell Anemia

Fever	24
Joint Involvement	16
URI symp.	14
Vomiting	13
Abdominal pain	10
Headache	10
Cough	8
Anorexia	6
Irritability	6
Pallor	6
Diarrhea	5
Jaundice	4

A review of the presenting symptoms demonstrates the diversified nature of sickle cell anemia. The most frequently encountered were fever, joint involvement, symptoms of an upper respiratory infection, vomiting, abdominal pain, and headache. It should be noted that all of the patients with joint involvement had pain but, in addition, five of these patients had associated joint swelling. Cough, anorexia, irritability, pallor, diarrhea, jaun-

dice, and drowsiness were less frequently noted. The least frequently encountered symptoms were chest pain, abdominal distention, swelling of the face, dyspnea, epistaxis, hemiplegia, ulceration of the skin, convulsion, symptoms of genito-urinary tract infection, and swollen glands. Five patients were asymptomatic at the onset but on routine blood studies were found to have sickle cell anemia. The mother of one patient first brought her child to the hospital because she had noticed a large mass in the abdomen which on examination was found to be an enlarged spleen.

Symptoms in 220 Admissions

Abd. pain	62	Chest pain	10
URI symp.	62	Epistaxis	10
Joint pain	43	Jaundice	10
Pallor	40	Swoln. jnts.	8
Fever	40	Bl. trans.	8
Malaise, weakness, etc.	39	Dyspnea	7
Vomiting	32	Nausea	7
Headache	28	Diarrhea	6
Anorexia	24	Chills	5
Back pain	15	Constipation	5

The overall symptomatic picture of all admissions has been arranged in order of frequency. The most outstanding features were abdominal pain, symptoms of an upper respiratory infection, joint pain, pallor, high fever, generalized malaise, vomiting, headache, anorexia, and back pain. Less common features were: adenopathy, genito-urinary infection, retardation, distention, swelling of the face, fainting, skin ulcers, ear ache, hemiplegia, abdominal mass, and convulsion.

Physical Findings on 220 Admissions

Abd. Tend.	83	Pneumonia	19
Splenomegaly	74	Tender jnts.	10
Hemic Murmurs	71	Adenopathy	10
Hepatomegaly	63	Otitis Media	9
Pallor	51	Abd. Distention	8
Cardiomegaly	47	Epistaxis	4
U.R.I.	41	Dehydration	4
Jaundice	32	Skin ulcer	3
Fever	29	Dyspnea	3

Of the most frequently encountered physical findings, the more interesting were those of abdominal tenderness which was usually generalized, splenomegaly noted in twenty-six patients but palpated on seventy-four admissions, hemic murmurs, hepatomegaly, and pallor. Less common physical features included dyspnea, stiff neck or back, impetigo, and atelectasis.

Incidental but uncommon findings included rachitic rosary, hydrocele, alopecia areata, pyelonephritis, angioneurotic edema, fractured skull, branchial cleft sinus, and chicken pox.

The number of the signs and symptoms present is proportional to the severity of the crisis. The pathophysiology as outlined here is thought by most men to be responsible for the entire symptom-complex of sickle cell crisis. The bizarre shape and rigidity of the erythrocytes causes stasis of capillary blood flow, blocking the capillaries. When the capillaries are thus obstructed, there is a retrograde spread to the arterioles and small arteries with resultant thrombosis and infarction. When thrombosis occurs, coagulating substances such as thrombin and thromboplastin are liberated which further enhance thrombosis and precipitation of more cells out of circulation. The generalized vascular blockage produces anoxia which causes more cells to sickle. A vicious cycle is set up: sickling→ stasis→ anoxia→ further sickling. During a crisis, this cycle may occur anywhere in the body.

The laboratory findings, similar to the symptoms and signs, vary with the severity of the crisis. During a crisis there may be a rapid fall in the hemoglobin content and the red blood cell count due to hemolysis of the sickled cells. Immature red blood cells appear in the peripheral smear. The white count becomes elevated and may reach a level as high as 100,000 per cubic millimeter. There is usually a rise in the icterus index. Blood studies in the fifty-eight cases reviewed showed an average admission hemoglobin of 6.79 grams, a red blood count of 2,320,000 and a white count of 21,000. The hemoglobin on discharge from the hospital averaged 10.3 grams, the red blood count 3,040,000 and the white count 10,690.

Nearly all patients with sickle cell anemia have minor crises which require no immediate therapy. However, in this study 116 of 220 admissions were for definite hemolytic crises. Fifty-nine or 53.4 per cent of the 116 crises showed some evidence of infection. In fifty-seven of 116 crises there was no evidence of infection. Tonsillitis was found on twenty-eight occasions. Pneumonia occurred fifteen times, nasopharyngitis nine, tonsillitis and otitis two, and impetigo, measles, chicken pox, and urinary infection each one time. There was one case in which sickle cell crises coincided with the onset of acute rheumatic fever which progressed to rheumatic heart diseaes.

Sickle cell anemia must be differentiated from many diseases, but perhaps the most important are: rheumatic fever, and abdominal surgical emergencies. Because of the diversified nature of this disease and the resemblance of its symptoms to other entities, sickle cell preparations have become routine procedures on our colored pediatric wards. It should be remembered that the arthritic pains of rheumatic fever are more strictly limited to the joints and are more readily relieved by salicylates than are those of sickle cell anemia.

The abdominal crises, one of the most striking features of the disease, is responsible for more hospital admissions than any other complaint. The distinction between the abdominal manifestations of sickle cell anemia and those of an acute surgical disease is of the greatest importance. An unnecessary laparotomy may be extremely dangerous during the hemolytic crisis while, on the other hand, failure to explore the abdomen in a surgically amenable condition, such as acute appendicitis, may be just as tragic. In this series, only one patient had a laparotomy because of a mistaken diagnosis.

Some of the complications besides hemolytic rises are: cardiomegaly with hemic murmurs, skin ulcers, bone changes, and cerebral manifestations. Approximately one-third of our cases had cardiac enlargement with hemic murmurs. Cardiac changes in this anemia are more marked than in any other anemia because of the long standing anoxia with compensatory hypertrophy and dilatation of the myocardium. Central nervous system involvement occurs most frequently in the older patients and when present the prognosis is usually poor. Two of our patients had findings referable to the central nervous system. One had a hemiplegia with partial recovery and the other had an uncomplicated convulsive seizure. Both were in the older age group. One patient in this series had a skin ulcer of the leg which was refractory to treatment and required grafting several times. The course of this ulcer is rather typical, as ulcers associated with sickle cell anemia are usually very resistant to therapy.

In the past ten years there have been only two deaths in this hospital due to sickle cell anemia. One was a three year old colored male who was dead on arrival at the hospital. Post mortem blood showed 80 per cent sickling. The other was a twelve year old colored female who previously had a splenectomy at another hospital. She was admitted in crisis and died five and one-half hours later. Other than these two deaths a search of our autopsy files reveals only two additional deaths due to sickle cell anemia.

The treatment of the "sickler" has always been a problem and is still most unsatisfactory. Liver, iron, transfusions, sedation, and general supportive therapy are helpful in crises, but these measures do not prevent future crises. A total of 525 transfusions were given to this group. This is an average of 9.5 transfusions per patient. One patient received as many as 85 transfusions.

The role of splenectomy in the treatment of sickle cell anemia has not been clearly determined. The few available reports on operated patients indicate, in general, that the state of anemia has not been appreciably altered. However, in some of these patients hemolytic crises have ceased or diminished in frequency following removal of the spleen.

In the winter of 1948–49 the plight of several patients on the wards and

clinics of Children's Hospital with severe and recurrent crises of sickle cell anemia stimulated a renewed interest in splenectomy as a procedure which might be of benefit. During the first eight months of 1949 six patients with repeated hemolytic crises and splenomegaly were operated. Post-operative observations of these children vary from three to eleven months and average seven months. Five of the six patients have been entirely well without recurrence of crises or need for transfusions. Each of these has maintained a moderate anemia in the neighborhood of 8 grams hemoglobin and 2,500,000 to 3,000,000 red blood cells. The sixth patient remained asymptomatic for six months but in the subsequent four months has become severely anemic and required transfusion on two occasions. While the results to date are encouraging, the short post-operative observation period precludes a valid appraisal of splenectomy as a therapeutic agent in sickle cell anemia.

In conclusion a review of fifty-eight cases of sickle cell anemia occurring over a representative ten year period has been presented. Emphasis has been placed on the presenting symptoms, the over-all symptomatology, the physical and laboratory findings. The importance of differentiating sickle cell crisis from acute abdominal emergencies and rheumatic fever is again stressed. The unsatisfactory results obtained from present therapeutic measures has been pointed out. Finally, a brief review of the splenectomized patients at this hospital indicates that in certain selected cases of sickle cell anemia this may prove a beneficial procedure.

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CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: Francis J. Troendle, M.D.
William M. Crowell, M.D.
By Invitation: Vasilios Lambros, M.D.

Francis J. Troendle, M.D.

J. W., a seven year old white male was admitted to Children's Hospital because of listlessness, headache, and vomiting of two weeks' duration.

Two weeks before admission, the patient developed a sore throat, vomited several times, and was noted to be "sluggish" and drowsy. He was seen by a physician who stated that he had a pharyngitis and prescribed a course of oral "sulfa" and penicillin. The sore throat was relieved in a few days but the vomiting, drowsiness, and listlessness persisted. In addition, the child ate very little, complained of abdominal pains, and was constipated. He had an intermittent headache of moderate severity throughout his illness. His gums bled on several occasions. There was no history of fever or rash and there were no urinary symptoms. The child lost 4 to 5 pounds in weight in two weeks.

The past medical history revealed only that the patient had a tonsillectomy and adenoidectomy two and one-half months before his present illness. The parents stated that the boy was a "sensitive type" and felt that some of his symptoms might be psychogenic in origin. His vitamin and dietary intake was adequate.

The family history revealed no history of tuberculosis. The patient's father stated that he himself bleeds for two or three days following a tooth extraction but there is no familial history of a similar condition.

Physical examination on admission revealed a thin, pale, dehydrated, semi-stuporous white boy who could be aroused only with difficulty. His temperature was 98.0 F., pulse 48 per minute, and blood pressure 100/66. The respirations were slow and shallow. The eyes were normal and the fundi were not examined at that time. The ear drums were retracted but not injected. The gums were swollen; the tongue was smooth and "beefy" red; and the throat was inflamed. The neck was freely flexible. Heart, lungs, and abdomen were normal and there was no lymphadenopathy. Except for the depressed sensorium there were no positive neurological findings.

A blood count revealed a hemoglobin of 16.5 grams; erythrocytes were 5,230,000; leukocytes 7,200 with 71 per cent polymorphonuclears. Urinalysis was negative. A lumbar puncture was performed and clear, colorless fluid was withdrawn under apparently increased pressure. The fluid con-

tained 7 leukocytes, 10 milligrams per cent protein, 50 milligrams per cent sugar. No organisms (including acid-fast) were seen on direct smear and the culture was sterile. Patch test and 0.1 milligram O. T. were negative. A flat x-ray plate of the abdomen disclosed no abnormalities.

The patient was given ample parenteral fluids and a blood transfusion. He remained in semi-coma, refused most oral nourishment, and vomited frequently. On the fifth and sixth hospital days his blood pressure rose to 120/90; his pulse was still slow (40–70 per minute); and there was early bilateral papilledema. On the same day the patient had a few mild tonic convulsions, the pupils dilated, the right larger than the left; the corneal reflexes disappeared, and all other reflexes were depressed. A second spinal tap revealed 6 cells, 20 milligrams per cent protein, 50 milligrams per cent sugar, and 760 milligrams per cent chlorides. He died on the sixth hospital day.

DISCUSSION

Vasilios Lambros, M.D.: In handling a neurological problem, one should have a logical system to follow. Whenever a problem such as this is presented, a simple outline can help in formulating a diagnosis. As you know, the resulting symptomatology in neurological diseases is usually the same, yet the causative factors are different. The outline is as follows:

1. Congenital Anomalies
2. Degenerative Diseases
3. Trauma
4. Psychogenic Disorders
5. Infections
6. Neoplasms.

By considering the present case, several categories can be excluded.

The majority of congenital anomalies will be evident before the age of two years. Degenerative diseases are manifested around the age of two to four years. From the history it is apparent that the mental and physical development of this child was within normal limits.

No evidence or history of trauma was given and thus this can be excluded.

In spite of the history of emotional difficulties there is no psychogenic disorder of childhood which will produce a bradycardia and pulse of 40.

Careful consideration has to be given to infections. An encephalitis on a bacterial or virus basis can be excluded by the normal constituents of the cerebro-spinal fluid. The sore throat of which this lad complained was probably based on dehydration and malnutrition. The history of a tonsillectomy two months prior to the present illness may be significant in this case. The venous drainage from the tonsillar area to the brain may easily transmit a metastatic infection.

With the history of emotional disturbances, listlessness, malnutrition, and a pulse of 40, a neoplasm in the subcortical frontal area is the most likely possibility. It should be remembered that a slow expanding mass can gradually compress the brain substance without marked alteration in function until the balance is broken when a plethora of signs and symptoms occurs.

A fundoscopic examination initially is not recorded. It probably would have established a lead in the diagnosis. In the presence of papilledema a spinal puncture should be carried out with extreme caution or not performed at all in view of the possibility of upsetting the pressure equilibrium which has been established and thus precipitating symptoms which may lead to the patient's death.

In this case my diagnosis is an expanding tumor in the frontal area, most likely a glioma, but I cannot exclude an encapsulated abscess.

PATHOLOGICAL DISCUSSION

E. Clarence Rice, M.D.: Some of those present will recall that at one of our recent meetings, we discussed the illness of a patient who had meningitis, and at death a brain tumor was found. The case under discussion today is rather similar in that the diagnosis appears to lie between brain tumor and brain abscess. Prior to the availability of chemotherapy and the use of antibiotics, brain abscess was seen more frequently at this hospital than brain tumor. Since the use of these chemotherapeutic agents, the former is rarely seen. On the basis of probability then, brain tumor would be the most likely diagnosis.

When the patient came to necropsy the body was found to be poorly developed and nourished with some discoloration of the skin over the thorax. The examination was limited to the head.

The calvarium was thinner than usual in both parietal regions.

The brain weighed 1620 grams (Normal 1263 grams). When the dura was incised the underlying cortex herniated through. Two small protuberances beneath the dura in the parietal regions of the brain corresponded to the thinned out portions of the calverium. On removing the dura, the convolutional markings were found to have nearly disappeared due to increased intracranial pressure. The surface had a dull, slightly yellow color. After removal from the cranium the brain expanded.

Examination after fixation disclosed thrombosis of one of the dural veins in the right frontal region as it entered the longitudinal sinus. The thrombosis did not completely obstruct the large vessel. A marked difference was noted between the gyri of the two hemispheres. Those on the right were flattened as by edema. Those on the left were also flattened but had a macrogyric appearance except over the occipital lobe.

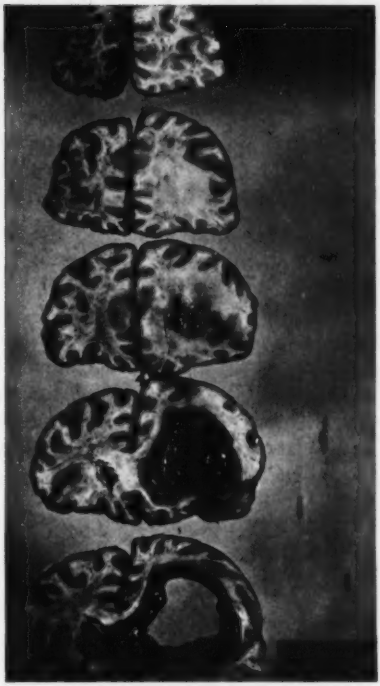

FIG. 1. Coronal sections of the brain showing marked flattening of the gyri and the abscess lying in the posterior left frontal lobe.

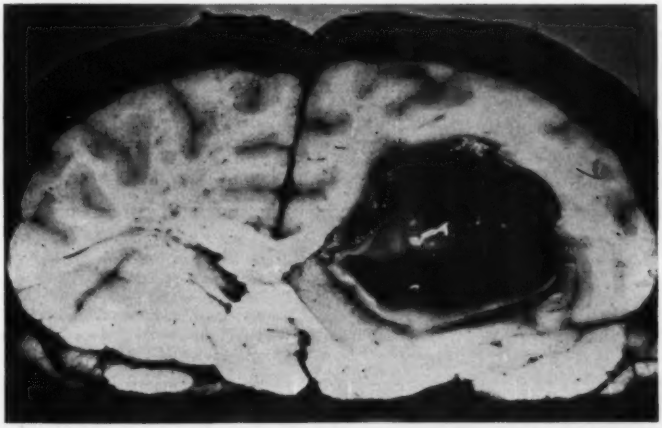

FIG. 2. Coronal section shows the massive abscess in the left hemisphere causing compression and displacement of the ventricular system to the right.

Coronal sections revealed marked flattening of the gyri on the left and in the posterior left frontal lobe, an abscess measuring 6.5 by 5.5 by 4.5 centimeters was found. It contained approximately 45 milliliters of greenish, foul smelling pus. The abscess was surrounded by a fairly well developed wall. The ventricles were displaced to the right as was the aqueduct. The white matter exhibited a defensive reaction which extended to the frontal pole. The entire brain was edematous, being most marked in the left cerebrum.

Microscopic examination disclosed the inner layer of the abscess wall to be made up largely of polymorphonuclear leukocytes, plasma cells, macrophages, and cellular debris. Adjacent to this, an increase in the number and size of the blood vessels plus marked proliferation of connective tissue along with a few astrocytes were seen. Externally to this, lymphocytic infiltration and numerous blood vessels were present. The adjacent brain tissue showed some rarification of the myelin sheaths and pyknosis of the nerve cells. The abscess appeared well localized without any break-through or spreading.

An anaerobic streptococcus was cultured from the pus of the brain abscess.

Pathological diagnoses:

Thrombosis of dural vein entering longitudinal sinus.

Subcortical abscess, left fronto parietal lobe.

Massive cerebral edema.

In retrospect we believe that the brain abscess followed the tonsillectomy performed four months previously. It is interesting that the only two brain abscesses following tonsillectomy—this patient and another, occurred rather close together. The other patient was admitted during the poliomyelitis season about six weeks after a tonsillectomy and the diagnosis of poliomyelitis was entertained until the necropsy gave the correct one.

These complications of tonsillectomy fortunately do not occur frequently and so far as I know, they represent the only complications of this type which we have seen in over 14,000 tonsillectomies. The routine use of the sulfonamides or a suitable antibiotic before and after such operations or the extraction of an abscessed tooth will make the likelihood of these complications remote and will help to reduce the morbidity following them.